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News from the CCWHC

Wildlife Technician - Western/Northern Regional Centre

Marnie Paskaruk graduated from the University of Saskatchewan in 1998 with a B.Sc. (Hons.) in Land Use and Environmental Studies/Biology. She accepted a term position with CCWHC in June 1998, as a wildlife technician on the avian botulism project. The need for a technician in W/N region increased, and the position became permanent. Marnie has done field and lab work on avian botulism since 1998, including radio-tracking of birds, bird banding, mortality surveys, and mapping outbreaks using Arcview. She also has worked on disease in amphibians, and surveillance for Chronic Wasting Disease. Marnie's background in biology and GIS, and enthusiasm for her work have been a real asset to the CCWHC. She can be reached at (306) 966-5815, or by email.

Feature Articles

The Research Group for Arctic Parasitology (RGAP): Heading North with CCWHC

Wild species are vital components of northern ecosystems and are very important as food for northern residents, for commercial and sport hunting and for tourism, all of which benefit the northern economy. Knowledge of factors affecting health of these animals, as individuals and populations, is increasing, but there is still much to learn. Accelerating changes in the northern environment, e.g., from global warming and resource development, give urgency to the need for baseline data on disease in arctic wildlife.

Since 1993, an international, interdisciplinary team based at the Western College of Veterinary Medicine (WCVM) has investigated parasites in northern ruminants and carnivores. The work focussed initially on muskoxen but now includes thinhorn sheep,

caribou, moose, mountain goats, bison, grizzly bears and lynx. The team includes parasitologists, biologists, veterinarians and others from Canada and the USA. On 1 January, 2002, the group was formally established within the CCWHC as the Research Group for Arctic Parasitology (RGAP), whose primary goal is to contribute to comprehensive understanding of animal parasitism in the Arctic and Sub-Arctic, with emphasis on the Nearctic.

The initial stimulus for this work was discovery in the late 1980s, by Anne Gunn (Resources, Wildlife and Economic Development (DRWED), Government of the NT) and Gary Wobeser (CCWHC W/N Region), of an unusual lung nematode in muskoxen. With involvement of Eric Hoberg, a parasite systematist and biogeographer with the US Department of Agriculture (USDA), this parasite was described as a new genus and species, Umingmakstrongylus pallikuukensis. Further research on U. pallikuukensis, facilitated by John Nishi, DRWED, Kugluktuk, formed the basis of the PhD thesis for Susan Kutz. Subsequent research on muskoxen has resulted in description of a new species of abomasal nematode, Teladorsagia boreoarcticus, and new geographic and host records for the lung nematode Protostrongylus stilesi. As part of a project led by John Nagy, DRWED, Inuvik, RGAP is investigating abomasal nematodes in muskoxen on Banks Island, NT, and their role in host population health.

In 1998, in collaboration with Alasdair Vetch, DRWED, Norman Wells, and Brett Elkin, DRWED, Yellowknife, research was initiated on parasites of Dall's sheep in the Mackenzie Mountains, resulting in new geographic and host records for two protostrongylid nematodes (Protostrongylus stilesi, Parelaphostrongylus odocoilei) affecting lungs of the sheep. In 1999, Emily Jenkins joined the team as a PhD student to investigate the distribution, epidemiology and effects of P. odocoilei in thinhorn sheep. This project includes experimental infection of captive thinhorn sheep at the WCVM, experimental epidemiology of the parasite in gastropods in the Mackenzie Mountains, and a survey of parasites of Dall's, Stone's and Fannin's sheep in the NT, the Yukon, northern BC and, on a limited scale, in Alaska. The survey is based on examination of fecal samples and, where possible, viscera or carcasses of hunter-killed and other sheep. Results are being compared with those from the Simmons Collection (held at the Canadian Museum of Nature in Ottawa), a unique assemblage of specimens and data from > 100 Dall's sheep collected in the Mackenzie Mountains in 1971-72 by N. Simmons and others from the Canadian Wildlife Service. This research is supported primarily by the Arctic Archival Observatory, a program at the University of Alaska Fairbanks funded by the US National Science Foundation (NSF).

Other current projects include parasite surveillance (based on fecal examinations) in a variety of northern species, and serosurveys for Toxoplasma in caribou, muskoxen and lynx, in collaboration with JP Dubey (USDA). A detailed survey of parasites of trapped lynx from the NT was initiated in 2001. Beginning in 2000, Eric Hoberg, Susan Kutz, and Brent Wagner (WCVM), have worked as field parasitologists for the Beringian Coevolution Project (BCP), an international NSF-funded study, for which Dr. Hoberg is a co-principal investigator, of the historical biogeography and evolutionary history of host-

parasite systems among a diverse range of mammals across Beringia from eastern Siberia through Alaska into north-western Canada.

RGAP strives to facilitate exchange of information about northern parasitology among researchers and northern residents, to be a source of expertise and training for parasitological investigations across the north, and to track developments in arctic and northern parasitology. Through its projects, residents of northern communities, as well as students in Saskatoon, have been educated in field and laboratory techniques related to wildlife parasitology. A short course in wildlife pathology and parasitology was presented by Trent Bollinger, CCWHC, Eric Hoberg, Brent Wagner and Lydden Polley for approximately 30 wildlife officers, technicians and biologists in Fort Smith, NT, in April, 2000. The first International Workshop on Arctic Parasitology (IWAP), held in Prince Albert National Park in October, 2000, attracted 45 participants from eight countries. IWAP 2 is tentatively scheduled for 2003 in Greenland. It is hoped that the educational role of RGAP, particularly in the north, will continue to grow.

Activities of RGAP involve collaboration among many individuals and agencies and depend on support from a variety of sources in Canada and the USA. Staff of government agencies and other groups concerned with wildlife management in the NT, Nunavut, the Yukon, BC, and Alaska have greatly facilitated RGAP activities. The Agriculture Research Service, USDA, through the Parasite Biology, Epidemiology and Systematics Laboratory, and especially the US National Parasite Collection, has provided scientific and organizational support for the creation and organization of RGAP. A vital component of this collaboration is an integrated approach to systematics, coevolution and historical biogeography of gastro-intestinal and respiratory nematodes of ruminants aimed at understanding the structure of the Holarctic parasite fauna. Support for field work in the Arctic is provided by residents of many northern communities, and hunters' and trappers' associations. Laboratory work at the WCVM is facilitated by colleagues from several areas of the university.

Financial support for RGAP has come from wildlife management agencies, co-management boards, and groups, including Foundation for North American Wild Sheep, Wild Sheep Society of BC, Canadian Wildlife Federation, Desert Bighorn Society, Safari Club International, Association of Mackenzie Mountain Outfitters, Department of Indian Affairs and Northern Development, WCVM Wildlife Health Fund, Arctic Institute of North America, and major pharmaceutical companies in Canada. Graduate and summer students are supported by WCVM interprovincial funds.

Further information about RGAP, including the mission statement, membership, publications and presentations, and IWAP proceedings, is available at the CCWHC website (wildlife.usask.ca) under satellite pages RGAP and IWAP, or from Susan Kutz, Lydden Polley or Brett Elkin.

Enhanced Passive Surveillance for West Nile Virus Infection in Wild Birds in Canada - 2001

The CCWHC, in collaboration with provincial and local public health authorities, organized collection of wild birds for West Nile Virus (WNV) surveillance in Canada, and submitted specimens for WNV detection to the Health Canada Special Pathogens and Zoonoses Laboratory, Winnipeg. It also organized collection of sightings of dead birds from the public, and maintained a data base for mapping and epidemiologic analysis. Surveillance focussed on corvids, since mortality in crows and blue jays is an early signal of local WNV activity. CCWHC accessioned 3890 bird carcasses (2796 submissions to Health Canada), and 2927 sightings (6817 cases overall): 3851 crows (1947 carcasses/1904 sightings); 2202 blue jays (1402/800); 129 ravens (85/44); 30 black-billed magpies (17/13); 10 gray jay sightings; and 595 other species (439/156). Ontario/Nunavut Region handled over 2/3rds of the workload.

A total of 126 WNV positive birds was reported in southern Ontario, from Windsor to York and Durham regions north and east of Toronto. Date of first pickup of a WNV positive bird, the species, and the total WNV positive birds in each health unit were: Windsor-Essex, 8 Aug., crow, 20; Halton, 13 Aug., blue jay, 7; Peel, 14 Aug., crow & blue jay, 17; Toronto, 15 Aug., crow, 41; Hamilton-Wentworth, 23 Aug., crow, 4; Chatham-Kent, 24 Aug., blue jay, 3; York, 25 Aug., blue jay, 23; Durham, 29 Aug., crow, 5; London-Middlesex, 29 Aug., crow, 3; Lambton, 30 Aug., crow, 1; Niagara, 11 Sept., blue jay, 1; Waterloo, 24 Sept., blue jay, 1. The last positive birds were picked up on 8/9 October.

In August, 21% of 190 Ontario crows tested were WNV positive, 31% of 151 (September), and 14% of 100 (October). The corresponding figures for blue jays were 3% of 196, 6% of 224, and 0.6% of 162.

Emergence of WNV in Ontario likely reflects the same factors, possibly dissemination of WNV with northward migrants in the spring, responsible for the shift in WNV distribution seen in the USA. Surveillance for WNV in wild birds represented an excellent example of cooperation among public health and other agencies at all three levels of government, and a national NGO such as the CCWHC. (Ian Barker, CCWHC Ontario/Nunavut Region).

Foot and Mouth Disease and Canadian Wildlife

The recent outbreak of Foot and Mouth Disease (FMD) in Europe evoked the spectre of its spread to North America and sparked many questions about what might happen should it arrive in Canada. No useful information has been reported about the effects of the current European outbreak on European wild animals nor is there any substantial literature about FMD in North American wildlife from outbreaks that have occurred in the past. Yet there is legitimate concern that the disease, or a national response to the

disease, might harm wildlife and economies dependent on wildlife if FMD were to turn up in Canada.

The usual reason for talking about wild animals in the context of FMD is that wild animals might become infected and then serve as reservoirs of virus and sources of new infections for domestic livestock during an outbreak. But wild animals also can be harmed by FMD. They can experience morbidity and mortality, reproductive loss and increased predation. Wild animals also can be harmed by disease control activities when these include killing, confinement, barricades or harassment of wildlife. Thus, wild animals must be viewed, not just as potential sources of disease for livestock, but also as potential victims of both the disease and its control measures.

What is known about FMD in Canadian wildlife? Based on experience world-wide, it must be assumed that all wild Artiodactyls (cloven-hoofed animals) can become infected. Seven of Canada's 12 species of wild artiodactyls have been infected with FMD somewhere in the world: white-tailed deer, mule/black-tailed deer, elk/wapiti, feral pigs, bison, moose and caribou/reindeer.[1] It is almost certain that the other 5 native species and subspecies – mountain goat, bighorn and stone - dall's sheep, muskox, and pronghorn – also can become infected. Very little, beyond potential for infection, is known about FMD in these Canadian species.

White-tailed deer infected experimentally with FMD suffered typical clinical disease, and shed virus for up to 11 weeks after infection, thus remaining a source of infection for other animals for almost 3 months.[2] The geographic range of white-tailed deer in Canada extends from southern Labrador to southeast British Columbia, including all areas of major livestock production except the Fraser Valley of BC. The species is abundant over most of its range: e.g. about 350,000 in Ontario, 250,000 in Saskatchewan.

Populations of feral pigs exist in Canada, but their number, distribution and biology are not well defined. These animals, often imported European wild boar, have escaped from farms and have formed self-sustaining, reproducing populations. A population estimated at about 500 pigs is present in Manitoba, for example. Pigs infected with FMD shed very large quantities of virus and produce aerosols of virus that can be infectious to other susceptible animals over distances of several kilometres. A lot of study and thought has been given to how FMD might spread in populations of feral pigs, based on extensive studies of their population biology. Australian scientists estimated that FMD will spread among feral pigs at a rate of about 2.8 Km per day when pigs are at fairly low population density (1-2/ km2).[3]

No information is available to permit a reliable estimate of the effect FMD might have on wildlife populations in Canada. However, it seems very likely that infection could persist for long periods, perhaps permanently, among white-tailed deer. The potential for other species such as wild sheep and bison to maintain the infection for long periods also may be high. Whatever effect FMD might have on populations of these species, persistence of the virus within their populations would impose an immense economic cost on Canada's livestock industry, because of closure of export markets.

During outbreaks of FMD in livestock over the past 80 years, veterinary authorities appear most often to have selected one of two different options regarding how wild animals should be treated: to kill wildlife, i.e. to include all susceptible wild animals in eradication programs and attempt to kill them all in the eradication area, or simply to ignore wildlife when planning and implementing FMD control plans. There are problems with both approaches. Killing enough wild animals to achieve eradication usually is not possible for purely logistical reasons. In addition, the economic value of wildlife in terms of direct consumer expenditures, background value for general tourism, and subsistence and cultural values is about the same as that of animal agriculture and its associated economies. Thus, today's society is unlikely to tolerate large-scale sacrifice of wildlife in support of agribusiness.

But, simply to ignore wildlife in FMD control and response planning in Canada is perilous. With 12 species of susceptible wild animals, many of which are expanding in number and range, and many of which share habitat with livestock, infection of Canadian wildlife with FMD represents a terrible risk for animal agriculture, and vigorous steps are needed to reduce this risk as far as possible.

Realistic planning for responding to foreign animal disease incursions must accord to wildlife the same degree of thought and attention that traditionally has been given only to livestock. This requires substantial activity on three fronts. 1) Complete information about the populations of the 12 susceptible wild animal species in Canada must be gathered together in one place, mapped, placed on-line and kept up to date. These are the essential data for response planning. No such assemblage of data for these species in Canada currently exists. 2) A consultative and planning network of professional wildlife and veterinary scientists must be established. Planning and implementation of a foreign animal disease response that includes wildlife, as such plans must, absolutely requires the participation of Canada's professional wildlife biologists. This is a network that does not exist; it must be built from scratch and built right now. 3) The wildlife response network must be deployed immediately to develop consensus, in advance, on the range of acceptable methodologies that can be used to reduce the risk of transmission of disease from livestock to wildlife in the event of an foreign animal disease outbreak, or to limit or prevent its spread among wild animals. These methods will be drawn primarily from among the methods of modern wildlife biology. This network also should establish the conditions and species for which the various methods would be appropriate, and measures of their effectiveness.

These three actions are both feasible and affordable, and are absolutely essential. They will establish the necessary information, lines of communication, and science-based plans to achieve preparedness; anything less will not.

References: 1. United States Department of Agriculture. 1994. Foot and Mouth Disease: Sources of Outbreaks and Hazard Categorization of Modes of Virus Transmission. USDA:APHIS:VS, Centers for Epidemiology and Animal Health, Fort Collins. 38 pp.

2. McVicar, J.W. et al. 1974. Foot and mouth disease in white-tailed deer: clinical signs and transmission in the laboratory. Proc 78th Annual Meeting U.S. Animal Health Assoc. pp 169-180.

3. Pech, R.P. and J.C. McIlroy. 1990. A model of the velocity of advance of foot and mouth disease in feral pigs. J Appl Ecol 27: 635-650

Raptor Tissue Collection

We have had a good response, particularly from western Canada, to the request for carcasses or tissues of great horned owls and red-tailed hawks as part of a collaborative study with the Canadian Wildlife Service of lead exposure in raptors. We still need better coverage of Quebec and the Atlantic provinces. If you have carcasses from these species that you are willing to send for this study, please contact either Doug Campbell at 519-823-8800 ext 4556 or 4662, dgcampbe@uoguelph.ca or Tana McDaniel by email at chelidra@yahoo.com to arrange for shipment of the carcasses. The study will pay any shipping costs involved.

Disease Updates

Atlantic Region

Barbiturate poisoning in a bald eagle

A juvenile bald eagle picked up alive in a field in late August was brought to a well known wildlife rehabilitation centre. This bird was thought to be thin and may have had difficulty flying because of weakness.

In captivity, it quickly started to eat a ration of fish and deer meat. After 2 days, it could reach high perches. Two days later, it was fed a black duck of unknown history that had been in a freezer for approximately 2 years but appeared well preserved. The eagle consumed the entire duck that day. In the evening, it showed signs of depression and was dead by nightfall. At necropsy, the eagle weighed 4.8 kg, was in good body condition, had a full crop and stomach, and no gross or microscopic lesions. A portion of the food present in its crop, weighing approximately 200 g, contained the barbiturate pentobarbital at a concentration of 1,700 ppm, for a total amount of 340 mg in that portion. A 5-kg eagle could easily be euthanized with 500 mg of pentobarbital injected intravenously. (At an average weight of 1 kg for the species, the black duck had been given an amount of barbiturate far in excess of what would have been needed to euthanize it.

Poisoning of bald that consumed carcasses of animals euthanized with barbiturates has been described by others. This particular incident emphasizes the ease with which this type of poisoning can occur. The managers of the rehabilitation centre are experienced people who are normally very diligent about the quality of the food they offer to birds under their care. It is very easy not to think of the potential presence of barbiturates in carcasses of unknown source. (Pierre-Yves Daoust, Atlantic Regional Centre).

Newcastle Disease in pigeons, Prince Edward Island

During summer 2001, several pigeons with neurologic signs were presented to the hospital or the necropsy laboratory of the Atlantic Veterinary College (AVC), University of Prince Edward Island (PEI). In a least one instance, four or five pigeons with neurologic signs had been observed in a backyard over a period of a few weeks. Of 18 birds submitted (the majority between June and September), at least 8 (4 adult, 4 immature) had definite neurologic signs, including circling, head tilt, and tremors. Suspicious signs in some other birds included inability to fly (without evidence of trauma) and a drooping wing. Gross lesions were rarely seen in these birds at necropsy. The main lesions identified microscopically included nonsuppurative encephalitis which most often was very mild, even in birds with marked neurologic signs, and a multifocal interstitial nephritis of moderate intensity. Two birds, both immature, had lesions suggestive of infection of their immune system by a circovirus (bursa of Fabricius in one bird, spleen in the other). This virus is known to cause immunodeficiency in infected pigeons. Specific tests for the presence of Newcastle Disease (ND) virus of pigeons (pigeon paramyxovirus-1) were done on samples from six birds. Immunohistochemical stains revealed the presence of the virus in the brain and/or kidneys of three of four birds. The virus was isolated from specific-pathogen-free chicken embryos inoculated with pooled samples of brain and kidneys from three (including the bird negative by immuno-histochemistry) of five birds. Serum samples from the two pigeons negative on culture had a positive antibody titre (1:64 dilution) to paramyxovirus by the hemagglutination inhibition assay, suggesting at least exposure to the virus.

Newcastle Disease, targeting primarily the nervous system, swept through pigeon flocks in continental Europe in the late 1970s and early 1980s. The origin of the disease may have been the Middle East. This epizootic spread to Britain in 1983 and to the northeastern United States in 1984. In Canada, the disease was identified in 1985 in flocks of racing pigeons in Ontario, Alberta and British Columbia. However, widespread use of inactivated ND virus vaccine in racing pigeons has contributed to a decline in its frequency. This pigeon-adapted strain of ND virus appears to have limited ability to spread to domestic poultry. Ingestion of virus-contamined feces is the most likely route of infection. Prominent clinical signs in affected pigeons include profuse green diarrhea followed by nervous signs such as lameness, drooping wings, incoordination and torticollis. Newscastle Disease can be a threat to birds of prey consuming infected pigeons.

Although still modest, the number of pigeons submitted from PEI to AVC in 2001 was higher than in the previous 5 years (average of 3.6 per year, range from 0 in 1997 to 10 in 2000). This could suggest that there was an unusually high incidence of ND in the province, this year. Alternatively, the increased submissions rate could have reflected enhanced awareness among the public about morbidity and mortality in wild birds, as a result of the West Nile Virus surveillance program in this region. Pigeons with similar neurologic signs have been submitted to AVC in previous years, including a cluster observed in one backyard in 1998. However, none of those cases was investigated for the presence of ND virus. Newcastle disease may have existed on PEI for many years, but with a fluctuating incidence that may be influenced by the proportion of the population that is susceptible to the disease in any given year, emergence of strains of the virus with differing virulence, and/or other factors. (Pierre-Yves Daoust and Scott McBurney, Atlantic Regional Centre).

Tuberculosis in a Thick-billed Murre

A thick-billed murre (Uria lomvia) was shot in early March 2001 on Long Island, Bonavista Bay (Newfoundland). The hunter was concerned because of the presence of a lump on the bird's chest. No lump was visible on gross post mortem examination; however, there were 10-15 irregularly shaped masses between the liver and sternum that looked like mycobacterial lesions (up to 1.5 cm diameter). These extended in a rough line that might have suggested some previous penetrating wound. Upon acid-fast staining (a special stain for mycobacteria) of sections of two separate masses, numerous acid-fast bacilli were seen. Follow-up culture through the Newfoundland public health laboratory identified the presence of Mycobacterium avium. Histologic examination of the masses at the Atlantic Veterinary College, UPEI, identified the presence of very large multinucleated giant cells typical of mycobacterial infection, and an acid-fast stain confirmed the presence of numerous small bacterial rods.

As avian mycobacteria are not commonly reported in free-living birds it is considered important to report this for the benefit of others who may be working in this field. Dr. Hugh Whitney, Provincial Veterinarian (Province of Newfoundland and Labrador).

Lyme Disease in Newfoundland

Newfoundland has a relatively small number of resident tick species. Though pets have been identified bringing ticks, such as Rhipicephalus sanguineus and Dermacentor variabilis, from mainland habitats, these have not been shown to establish permanent populations.

In recent years there have been reports of Ixodes scapularis found on dogs and people who have no history of travel. In most cases, a recent trip into the bush has been identified as the point where the tick was picked up.

Five such cases have been identified across Newfoundland (see map) and in one case the Health Canada laboratory in Winnipeg identified the presence of Borrelia burgdorferi (causative agent of Lyme Disease) in the tick. This tick was removed from a dog in Cape Broyle that had recently been in the woods behind the owner's house. The dog was given preventive treatment by the veterinary clinic, so there was no actual diagnosis of Lyme Disease in the animal.

The Newfoundland habitat is not considered to be one that is ideal for the proliferation of this arthropod; however, further research is being initiated into the distribution and abundance of this and other tick species. Further information on ticks in this province can be found at: www.gov.nf.ca/agric/pubfact/AnimalHealth.htm Dr. Hugh Whitney, Provincial Veterinarian, Province of Newfoundland and Labrador.

Quebec Region

Type C Botulism Outbreak on the Shore of the St Lawrence (First occurrence in Quebec?)

At the end of summer, 14 ducks including 4 Northern Pintails, 3 American Black Ducks, 4 Mallards, a Northern Shoveler, an American Wigeon, and a Green-winged Teal were submitted by the Canadian Wildlife Service to the Quebec Regional Centre, in Saint-Hyacinthe. Most of the birds were found during the second half of August in the Anse-du-fort of St-Pierre Lake, a wider portion of the St Lawrence seaway located about halfway between Montréal and Québec City used by many waterfowl during spring and fall migration. The others were found during early September at the waterfowl refuge of Baie-du-Febvre/Nicolet, in ponds created by Ducks Unlimited. The locations are shallow marshes with cattails, submerged vegetation and a sandy bottom. Nine birds were alive when submitted most were moribund but six had paresis or paralysis of the wings, the legs or neck . The birds were unremarkable grossly, except for variably diminished muscle mass along with empty proventriculus and gizzard. Although various histological changes were observed, no cause was found to explain the simultaneous illness and death of these birds. Possible causes of death considered included lead or pesticide poisoning and botulism. Liver lead values were within normal limits and pesticide screening performed on a pool of tissues was unrewarding. Type C botulism toxin was detected in two pools of serum from blood collected from the live birds, submitted to the Laboratoire de pathologie animale of the Ministère de l'Agriculture, des Pêcheries et de l'Alimentation du Québec (Sainte-Foy, Québec).

To our knowledge, type C botulism in waterfowl had not been officially diagnosed before in Québec. We could find no reference to detection of this disease in the province. During

a field survey by volunteers, an additional 17 Mallards, 9 Black Ducks, 6 Northern Pintails, 6 Green-winged Teal, 1 Blue-winged Teal, 1 Gull (unidentified), 1 Scaup, a few unidentified Sandpipers and >47 unidentified birds were found at the sites. It is estimated that 150-200 birds died during the outbreak. Interestingly, waterbirds were found dead in the same areas during the summer of 1999. No firm diagnosis was made at that time, because of the poor state of preservation of the birds. During the 1999 episode, approximately 50-100 ducks found dead in the Anse-du-Fort area. Waterfowl banding was performed in this location in 2000 but no mortality was noticed by wildlife biologists. The outbreaks in 1999 and this year coincided with a lowered water level within the marshes, which was not the case in 2000.

It is interesting to speculate if such events could recur in the future, considering the fluctuation of water levels observed within the St Lawrence Seaway for the last decade or so. Water level changes undoubtedly affect the wetland habitats found along the St Lawrence. Water level changes, in this case reduced levels, could concentrate aquatic invertebrates and kill them because of increased salinity or diminished oxygen levels within their environment. Degrading invertebrate carcasses might provide an anaerobic environment for replication of C. botulinum and toxigenesis. This situation should be monitored to prevent larger epidemics that could affect the valuable waterfowl resource. (André D. Dallaire, Québec Regional Centre. The professional collaboration of Dr Sébastien Monette in performing necropsies and histopathology on these birds is acknowledged and was greatly appreciated as was the collaboration of Mr Jean Rodrigue, biologist for the Canadian Wildlife Service - Québec region, in providing field information relating to these outbreaks.)

Ontario Region

Suspected cyanide poisoning in birds

Many plants, including those in the genus Prunus, which includes cherries and apples, have glycosides in leaves, twigs and seeds, that when digested release cyanide gas, which can lead to respiratory failure. Flesh of the fruit does not contain these glycosides. In September, 7 from a group of > 25 starlings found dead over a 2 day period in Hamilton were examined. No lesions were found, but all had substantial amounts of recently eaten cherries in their gizzard. This material released cyanide gas when crushed. Although other causes of death cannot be excluded, it is possible that these birds died of acute cyanide poisoning as the result of ingestion of the seeds. A number of ruffed grouse from different parts of the province that were examined had similar findings, in that their gizzards contained recently ingested cherries or choke cherries. Pits of these fruits were present in the gizzard, but none were evident in the lower digestive tract, suggesting that they were ground up during the digestive process, during which cyanide might have been released. All of the grouse had traumatic injuries. Speculatively, their ability to fly might have been impaired by the effects of cyanide.

Ethylene Glycol: Ethylene glycol (antifreeze) poisoning was identified as the cause of death of individual birds in two incidents. Diagnosis was based on identification of calcium oxalate crystals in renal tubules, accompanied by evidence of damage to the tubules. One case involved a blue jay from a suburban area. The second involved a turkey vulture. This was the third vulture reported dead in the area, but was the only one submitted for post-mortem.

Zinc Phosphide: Zinc phosphide is a rodenticide used in outdoor applications, such as protection of planted trees and shrubs. Poisoning with zinc phosphide has been diagnosed in Ontario previously in white-tailed deer and wild turkeys feeding in an orchard, and in Canada geese grazing on landscaped properties in an industrial park. A recent incident involved Canada geese in a new industrial park in the Ottawa area; 6 geese were found dead. (Doug Campbell CCWHC - Ontario/Nunavut Region; Barb Campbell (CWS).

Type E botulism, Lake Erie

Since 1999, botulism has occurred annually on Lake Erie, beginning with episodic mortalities in gulls and shorebirds during the summer, and reaching a peak during the migration of fish-eating birds, particularly common loons and red-breasted mergansers, in October and November. Source of the toxin is not known; many of the loons have fish remains in their gizzards. Identification of gizzard contents from the 1999 outbreak, done at the MNR laboratory in Wheatley, identified gobies (a species of fish from the Caspian Sea that has recently invaded the Great Lakes) as the species most commonly present. Since loons and mergansers eat live prey, it raises the question of whether sick gobies, affected by botulism themselves, are being consumed. Birds such as gulls are presumably ingesting toxin while scavenging on carcasses of fish, birds, or mudpuppies, that may have died of botulism or other causes. The geographic area of botulism occurrence has shifted from the western basin of Lake Erie in 1999 to the middle and eastern basins. This year, much of the reported mortality occurred in the area between Port Dover (east of Long Point) eastwards to Dunnville, at the mouth of the Grand River. Doug Campbell (CCWHC), Jeff Robinson (CWS), Tim Johnson, Phil Ryan (MNR)

Parvovirus in raccoons

Parvovirus has long been recognized as a potential cause of mortality in raccoons housed in captivity, but now appears to be developing as a common cause of death in free-ranging raccoons. This fall, raccoons with lesions of parvovirus have been seen from widely-scattered locations in Ontario. Additionally, there have been reports of mortality in raccoons that had the characteristics of parvovirus. Disease in raccoons is caused by a parvovirus indistinguishable from feline parvovirus (the cause of feline panleukopenia in cats), and results in severe, acute illness involving widespread destruction of intestinal epithelium, with death rapidly resulting from dehydration, electrolyte imbalance or

secondary sepsis. Raccoons dying of parvovirus are likely to be found dead, rather than walking about sick, as is often the case with canine distemper virus. Feline panleukopenia virus outbreaks have occurred at some animal shelters in southern Ontario this fall as well, and there may be some connection between the prevalence of this condition in cats and its occurrence in raccoons. (Doug Campbell, CCWHC).

Canine Distemper Virus in mustelids: Canine

Distemper Virus (CDV) has been diagnosed by immunohistochemistry, in pine marten (Martes americani) and fisher (Martes pennanti) during the past year. The marten, fitted with a radiotelemetry collar, was found dead during a population ecology study in the Red Lake district. The fisher was found dead in a live trap during the fall raccoon rabies trap-vaccinate-release program in eastern Ontario. CDV has been reported in marten previously, but no previous cases in fisher have been found. Both marten and fisher are semi-solitary, with relatively little contact between individuals, which raises the question of how the virus was transmitted. CDV is caused by a virus which does not survive well in the environment, and is generally transmitted by close (nose to nose) contact between affected and susceptible animals. (Doug Campbell (CCHWC), John Fryxell, Mark Andruskiw (Department of Zoology, University of Guelph), Rick Rosatte, Dennis Donovan (Rabies Research, MNR).

Western and Northern Regions

Newcastle Disease and Avian Cholera in Cormorants with out Epidemic Mortality

Both Newcastle disease virus (ND) and avian cholera (AC) (infection with the bacterium Pasteurella multicida) were detected in summer 2001 at nesting colonies of double-crested cormorants in Saskatchewan at which each disease has caused large-scale mortality in previous years. However, there was no die-off at any of the three locations in 2001. On 21 July, one dead and two sick half-grown cormorants were found on Island A, Doré Lake, a colony of several thousand birds. Highly pathogenic ND virus was isolated from two birds, each with microscopic lesions typical of ND. No significant mortality was detected on this colony during this or two subsequent visits. On 1 August, seven cormorants, a gadwall and a lesser scaup were found on a large cormorant nesting colony in Kazan Lake which experienced mortality of several thousand birds due to avian cholera in 2000. Pasteurella multocida was isolated in high numbers from the five cormorants examined and from both ducks. The colony was visited again on 28 August but no significant mortality or sickness was evident. On 9 August, 1 sick cormorant was collected from a large colony on Last Mountain Lake on which about 200 dead birds were noted. Highly pathogenic ND was isolated from this bird which also had typical microscopic lesions. No further large-scale mortality was reported from this colony.

At Doré and Kazan lakes, these diseases were detected only because the colonies were under surveillance and autopsies were done on the small number of birds found dead when the colonies were visited. At Last Mountain Lake, detection of the disease was similarly due to the chance submission of a specimen. These data show that ND and AC can affect birds on a colony without necessarily resulting in widespread disease. The factors that determine whether or not epidemic disease will occur when both the infectious agents and susceptible hosts are present remain to be discovered. Ruys Beaulieu - SERM, Ted Leighton, Gary Wobeser - CCWHC).

Polioencephalomalacia in Wild Ungulates

Polioencephalomalacia (PEM), from the Greek polios (grey), enkephalos (brain) and malakia (softening), describes a condition in which grey matter of the brain degenerates or is 'softened'. A disease of this type has been recognized in domestic cattle, sheep and goats for many years. Sporadic cases have been diagnosed in white-tailed deer and pronghorn antelope in Saskatchewan since 1974.

PEM is a metabolic disorder in which disruption of energy supply to nervous tissue results in the brain lesions. Two mechanisms have been proposed to explain the energy disruption. One hypothesis is that PEM results from deficiency of thiamine (vitamin B1), a critical cofactor in carbohydrate metabolism. Carnivores are dependent upon thiamine in their diet, and diets deficient in thiamine can result in brain lesions similar to PEM. Ruminants do not require dietary thiamine, because bacteria in the rumen synthesize this vitamin. Thus, deficiency seems less likely in ruminants. However, it has been suggested that alterations of rumen microflora might result in decreased thiamine production or destruction of thiamine in the rumen, leading to deficiency. PEM commonly occurs in animals eating carbohydrate-rich food, such as grain, and one thought is that this diet might disrupt rumen bacteria. Attempts to prove that animals with PEM are deficient in thiamine have been inconclusive. The second hypothesis is that PEM is related to a high dietary intake of sulfur. The proposed mechanism is that relatively non-toxic forms of sulfur in food or water are altered in the rumen by microbes to produce toxic sulfide ions and H2S that are absorbed and interfere with cellular energy metabolism Because the brain requires a continuous high level of energy production, it could be affected by a cellular toxin that inhibits cell respiration, resulting in energy deficiency. PEM occurs in cattle with high sulfur intake and has been reproduced by giving experimental animals large amounts of sulfates.

During 2001, six wild animals with PME have been submitted to the W/N Regional Centre. This is morecases than usual and, for the first time, moose were affected. All the animals came from grain-growing areas of the province. This reflects a recent marked increase in the occurrence of small groups of moose in agricultural regions far from any forest. Signs observed in the animals were typical of those in the wild ruminants with PEM that we have seen over the years, with animals unable to stand or falling down, blind and indifferent to their surroundings. Changes in the brain are subtle and there may

be no lesions visible grossly. Deer may have brain swelling, with displacement of the cerebellum into the foramen magnum.

Pronghorns often have hemorrhage within the brain. There were no gross lesions in the moose. In some animals, there may be areas of fluorescence when the brain is examined under U.V light. Microscopically, there is necrosis of cerebrocortical neurons, often in a laminar pattern, together with a spongy appearance of the neuropil.

The cause of PEM in wild ruminants is unknown but is likely similar to that proposed for domestic livestock. Much of the surface water available in southern Saskatchewan has elevated levels of sodium and magnesium sulfate. In drought years, such as 2001, the concentration of minerals in the remaining water is likely very elevated. Some plants may contain high concentrations of sulfur, so that animals may obtain sulfur from both food and water. All the animals examined in 2001 had grain in their rumen and this has been a common finding in the past. Animals on a high carbohydrate diet may have altered rumen microflora which could influence both sulfide production in the rumen and also potentially change thiamine production. (Gary Wobeser, Western/Northern Regional Centre)

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